Associations between Cardiometabolic Index and heart failure in adults aged ≥ 20 years: Evidence from the NHANES 1999–2018

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Abstract

Background: Heart failure (HF) is a prevalent global health issue with increasing incidence due to aging populations and advancements in treatment. The Cardiometabolic Index (CMI), a new marker combining waist-to-height ratio and the triglycerides-to-HDL cholesterol ratio, has shown promise in predicting cardiovascular risks. However, the relationship between CMI and HF remains unclear, warranting further investigation. This study aims to examine the association between CMI and HF to better understand and potentially identify HF risks.

Methods: This study included 101,316 participants, of whom 22,042 met the selection criteria to investigate the correlation between the CMI and heart failure. The CMI is calculated as the product of the waist-to-height ratio (WHtR) and the triglycerides-to-HDL cholesterol ratio (TG/HDL-C). Data collection involved personal interviews to gather heart failure information, with HF diagnosis based on specific questionnaire responses. Clinical and biochemical data encompassed a wide range of variables, including demographic details, health status, and biochemical markers. Statistical analyses leveraged complex survey design from the National Health and Nutrition Examination Survey (NHANES), using weighted regression and chi-square tests to compare groups and multivariate logistic regression to examine the CMI-HF relationship across adjusted models. An analysis of the threshold effect elucidated the nonlinear dynamics present between CMI and HF, incorporating subgroup analyses to investigate the interactions among variables and the Receiver Operating Characteristic (ROC) curve was employed to evaluate the diagnostic utility of CMI in comparison to Body Mass Index (BMI) for the detection of HF.

Results: In this study, based on specific inclusion and exclusion criteria, 706 individuals were diagnosed with HF, representing 3.2% of the total population. The findings indicated a significant association between elevated CMI levels and an increased risk of HF (OR = 1.13; 95% CI, 1.07–1.17, $p < 0.001$), with each unit increment in CMI level being associated with a 13% increase in HF risk. Subgroup analyses revealed the stability of the CMI-HF relationship across various subgroups, identifying race, history of heart disease, and hypertension status as key modulators of the strength and direction of the CMI-HF association. Moreover, smooth curve fitting and threshold effect analysis demonstrated a non-linear relationship between CMI and HF, with an inflection point at a CMI level of 6.49. Below this threshold, the incidence of HF increased with rising CMI levels. Additionally, the diagnostic capabilities of CMI and Body Mass Index (BMI) in identifying HF were compared, with the area under the curve (AUC) values for CMI surpassing those for BMI, indicating a superior ability of CMI in identifying HF.

Conclusion: Our research indicates that the level of CMI bears a substantial positive correlation with the incidence risk of HF, with the relationship between CMI and HF being non-linear. Additionally, the CMI is a better predictor of HF than the BMI.

1. Introduction

HF represents a substantial global health challenge, impacting in excess of 64 million individuals globally. HF is characterized as a multifaceted clinical syndrome, manifested through symptoms and signs arising from any structural or functional impairments that compromise the ventricular capacity for blood filling or ejection[1,2].

while the incidence of HF has stabilized or even declined in industrialized countries, the prevalence continues to increase due to population aging, improved treatment and survival rates for ischemic heart disease, and the availability of effective evidence-based therapies that prolong the lives of patients with HF. There are geographical variations in HF epidemiology, with a notable lack of data from developing countries where HF features differ from those observed in the Western world[2]. HF incurs substantial economic ramifications on a global scale, with the estimated total expenditure reaching $30.7 billion in the United States in 2012, a figure anticipated to escalate markedly by 2030[2–4]. Shifting focus to China, heart failure emerges as a formidable healthcare dilemma, as delineated by a comprehensive national assessment conducted in 2017. The age-adjusted prevalence and incidence rates were identified as 1.10% and 275 per 100,000 person-years, correspondingly, revealing the existence of approximately 12.1 million current cases and an annual increment of 3.0 million new cases among individuals aged 25 and above. The economic impact is significant, with per capita expenditures for inpatient and outpatient services recorded at $4406.8 and $892.3, respectively, and over 40% of patients necessitating recurrent hospital admissions within a single year[5]. Thus, HF represents a global public health challenge that necessitates improved preventive, diagnostic, and therapeutic strategies to address its social and economic burden.

The CMI is a new marker developed to discriminate diabetes mellitus, whose components are associated with the identification of coronary heart disease and metabolic syndrome. It is calculated as the product of the WHtR and the TG/HDL-C[6]. The WHtR is posited as a superior prognostic tool for cardiovascular risks compared to the traditional BMI and waist circumference metrics, primarily due to its incorporation of an individual's stature, thereby furnishing a more precise gauge of adipose tissue distribution. Furthermore, WHtR demonstrates a more robust correlation with coronary heart disease and cardiovascular risk determinants relative to waist circumference or BMI in isolation, thus enhancing its efficacy in the stratification of risk[7]. Concurrently, the TG/HDL-C ratio is acknowledged as a cogent marker for cardiovascular morbidity owing to its capacity to mirror the concurrent elevation of triglycerides and diminution of HDL-C, each an autonomous harbinger of cardiovascular pathology. An escalated TG/HDL-C ratio is invariably linked with augmented susceptibilities to insulin resistance, metabolic syndrome, and coronary arteriopathy[8,9]. This index is proposed because patients with type 2 diabetes often exhibit obesity and dyslipidemia, which significantly contribute to their risk of coronary atherosclerotic diseases and macrovascular dysfunction. While the prevalence of metabolic syndrome in the context of cardiovascular disease significantly hinges on its definition, demographic factors, ethnicity, and gender, it is widely acknowledged that individuals with this syndrome face a substantially elevated risk of cardiovascular diseases and heart failure[10]. However, the connection between CMI and HF remains ambiguous, including the potential dose-response relationship between them.

Given the aforementioned context, this study aims to evaluate the cross-sectional association between CMI and HF using data from the NHANES, and further ascertain the value of CMI in identifying HF.

2. Materials and methods

2.1 Study population

The NHANES, a project of the National Center for Health Statistics (NCHS), is a periodic survey that evaluates the health and nutritional status of Americans[11]. It operates on a biennial basis, with each session covering a unique cohort to reflect demographic shifts and allow trend analysis. The protocols of the NHANES have received approval from the ethics committee of the NCHS. The collected data are made available on the website of the Centers for Disease Control and Prevention (CDC) at https://www.cdc.gov/nchs/nhanes/.

In this study, 101316 participants from 1999 to 2018 were selected, and 22042 people were determined according to the screening criteria, in order to explore the relationship between CMI index and heart failure (Fig. 1). The exclusion criteria of this study were age<20, pregnancy, lack of HF status data, CMI, TG, HDL-C, WC, and height.

2.2 Clinical and biochemical data collection

In the NHANES survey, HF information was obtained through personal interviews using health questionnaires. The diagnosis of HF was based on the response to the "MCQ160B" question in the MCQ section, where a 'yes' answer to "Have you ever been told by a doctor or other health professional that you have HF?" indicated HF. This methodology has been referenced in several prior studies based on NHANES data[12–14].

This study also included age (years), gender (male/female), race (Mexican American/Other Hispanic/Non-Hispanic White/Non-Hispanic Black/Other race or multiracial), education level(less than 9th grade/9-11th grade/high school grad /some college/ college graduate or above), poverty income ratio (PIR), smoking status, height(cm), waist circumference (WC, cm), hypertension, diabetes, coronary heart disease, angina, heart attack, stroke, body mass index (BMI, kg/m^2), triglyceride (TG, mmol/L), total cholesterol (TC, mmol/L), high density lipoprotein cholesterol HDL-C(mmol/L), low density lipoprotein cholesterol (LDL-C, mmol/L). Detailed measurement procedures for all variables in this study are disclosed in the NHANES database.

Further calculate the relevant index:

WHtR = WC (cm)/ height (cm),

CMI = TG (mmol/L)/HDL-C (mmol/L) × WHtR.

2.3 Statistical analysis

All statistical analyses were conducted employing the NHANES sampling weights, duly accounting for the intricate, multistage, stratified survey design. The demographic and clinical characteristics of the study cohort were stratified into two distinct groups based upon the presence or absence of HF among the participants. Continuous variables were delineated as mean ± standard deviation (SD), whereas categorical variables were delineated as proportions. To ascertain disparities between the groups in terms of baseline characteristics, both continuous and categorical, weighted linear regression and weighted chi-square tests were respectively applied. The association between the CMI and HF was scrutinized through multivariate logistic regression analyses, structured into three distinct models: Model 1 (unadjusted), Model 2 (adjusted for gender, age, and ethnicity), and Model 3 (comprehensively adjusted for all gender, age, ethnicity, education level, family poverty income ratio, hypertension, diabetes, coronary heart disease, angina, heart attack, stroke, smoking status). The non-linear relationship between CMI and HF, including the determination of the inflection point, was elucidated using a threshold effect analysis approach, incorporating variable adjustments and smooth curve fitting techniques. Subgroup analyses were conducted to delineate the study population into various strata, including gender, ethnicity, age, educational attainment, hypertension, diabetes, coronary heart disease, angina, heart attack, stroke, and smoking status, with the introduction of interaction terms to examine heterogeneity across the subgroups. Finally, The ROC curves were constructed for the participants to calculate the AUC values, thereby facilitating the evaluation of the diagnostic superiority of CMI relative to BMI in identifying HF, with the AUC comparisons conducted via the DeLong test. All statistical computations were executed utilizing R (version 4.2.0) and EmpowerStats (version 6.0). $P < 0.05$ was considered statistically significant.

3. Results

3.1. Baseline characteristics

A total of 22,042 adults were recruited based on inclusion and exclusion criteria. Of the 22,042 subjects, 706 were diagnosed with HF, representing 3.2% of the total population. The mean age of total participants was 47.32 ± 18.03 years and included 10,862(49.41%) males and 11180(50.59%) females, of whom 69.06% were non-Hispanic white, 10.45% were non-Hispanic black, 8.18% were Mexican American, 5.61% were other Hispanic, and 6.69% were from other races. The mean BMI, height, and waist circumference were 28.67 ± 6.61 (kg/m2), 169.01 ± 10.05(cm), and 98.74 ± 16.04 (cm), respectively, and the mean CMI was 0.79 ± 1.08.

Table 1 delineates the clinical profiles of the study participants, employing HF as a key variable for column stratification. A notable disparity was observed in the demographic and fundamental clinical traits among patients with HF compared to those devoid of the condition. Specifically, individuals within the HF cohort were predominantly male, of advanced age, and more frequently identified as non-Hispanic black or white. They also tended to be smokers, possess lesser educational achievements, and report lower family income levels relative to the poverty threshold. Remarkably, HF patients exhibited elevated measures in BMI, waist circumference, TG, and CMI levels, whereas their direct total cholesterol, HDL, and

LDL cholesterol levels were diminished in comparison to their non-HF counterparts. Furthermore, the incidence of hypertension, diabetes, coronary artery disease, angina, heart attack, and stroke were significantly higher among HF patients, underscoring the interconnection between these conditions and the heightened risk of developing HF.

3.2. Association between CMI and HF

Our results suggest that a higher CMI is associated with the likelihood of an increased prevalence of HF (Table 2). Both our model 1 (unadjusted model) (OR = 1.15; 95% CI, 1.11–1.19, p < 0.001) and model 2(adjusted for gender, age, and race variables) indicated that this correlation was significant (OR = 1.17; 95% CI, 1.13–1.22, p < 0.001). The significant link between CMI and HF persisted in model 3 (additionally adjusted for education level, family PIR, hypertension, diabetes, coronary heart disease, angina pectoris, heart attack, stroke and smoking status) (OR = 1.13; 95% CI, 1.07–1.17, p < 0.001), indicating that each unit increase in CMI level was associated with a 13% increase in the risk of HF prevalence, respectively.

3.3. Subgroup analyses

To ascertain the stability of the association between CMI and HF across various subgroups, a subgroup analysis was conducted (Table 2). Interaction tests revealed statistically significant differences in the association between CMI and HF across subgroups(p < 0.05 for interactions) (Figure 2), defined by race (Mexican American/Other Hispanic/Non-Hispanic White/Non-Hispanic Black/Other race), history of heart attack (yes/no), and hypertension status (yes/no).This outcome suggests that racial background, hypertension status, and a history of heart attacks may serve as critical moderating factors, influencing the strength and direction of the relationship between CMI and HF. Conversely, gender (male/female), age (years), educational level (below high school/high school/above high school), diabetes status (yes/no), coronary artery disease status (yes/no), angina (yes/no), stroke (yes/no), and smoking status (yes/no) did not significantly impact this positive association (p > 0.05 for interactions).

3.4. Smooth curve fitting, threshold effect and saturation effect analyses between CMI and HF function

To further elucidate the relationship between CMI and HF, we analyzed both by smoothing curve fitting (Figure 3), threshold effect and saturation effect. We found a non-linear relationship between CMI and HF with an inflection point of 6.49. When CMI levels were below 6.49, the prevalence of HF increased with increasing CMI, with odds ratio (OR) was 1.26(95% CI =1.15–1.37, p < 0.001). When CMI reached the inflection point of 6.49, there was no significant correlation between CMI and HF (p = 0.665) (Table 3).

3.5. Accuracy of CMI and BMI for identifying HF

To compare the ability of CMI and BMI for identifying HF, we plotted the ROC curves of the two parameters (Figure 4) and calculated the corresponding optimal cut-off values, sensitivity, specificity and AUC values (Table 4). The results showed that the AUCs of the two parameters were all greater than 0.5, among them, CMI (AUC: 0.6056 and 0.6467) had the higher ability to recognize HF, followed by BMI (AUC:

0.5715 and 0.6139) (Delong P < 0.001). In addition, we calculated the optimal cut-off values of CMI and BMI for identifying HF as 0.566 and 27.145, respectively.

4. Discussion

In our comprehensive cross-sectional analysis involving 22,042 participants, we identified a positive correlation between CMI levels and the incidence of HF. To the best of our knowledge, this represents the first large-scale study to investigate the relationship between CMI and HF. Upon conducting further subgroup evaluations and interaction assessments, we discovered that this relationship persisted across most demographic groups, albeit moderated by factors such as racial background, the presence of hypertension, and a prior history of heart attack, which influenced both the strength and direction of the CMI-HF correlation. Intriguingly, our analysis unveiled a nonlinear relationship between CMI and HF, characterized by a critical inflection point at a CMI value of 6.49. Finally, employing ROC curves and the AUC values, we substantiated that CMI exhibits a superior discriminatory capacity for HF as compared to BMI.

Originating from the seminal work of Wakabayashi and Daimon[6], CMI integrates adiposity and lipid profiles for diabetes differentiation, leading to further exploration of its prognostic value in cardiovascular diseases by researchers like Guo and Liu, in relation to conditions like chronic kidney disease and the metabolically obese normal weight phenotype[15]. This work, including the insights from Zha et al., which highlight a nonlinear relationship between the CMI and the risk of diabetes[16], underscores its importance in identifying cardiometabolic risks, particularly in light of the role of diabetes in heart failure[17,18]. This nuanced association, particularly evidenced in a Japanese cohort study, sheds light on the intricate interplay between metabolic markers and cardiovascular outcomes[19].Our in-depth exploration of the association between the CMI and heart failure, demonstrating its utility in HF risk assessment, corresponds with discoveries such as those by Wang et al. regarding the impact of the CMI on cardiac structure, underscoring the essential role of CMI in evaluating the risk of heart failure[20].

In the smoothing curve fitting, below the threshold of 6.49, CMI emerged as an independent predictive factor for an elevated incidence of HF, highlighting its significance in identifying at-risk individuals. Notably, there is a "plateau phase", approximately from a CMI of 6.49 to 20, suggesting that within this interval, increases in CMI are not associated with a significant increase in heart failure incidence. In a study conducted by Lin et al., the WHtR, a constituent component of the CMI, and its correlation with cardiorespiratory fitness (CRF) were discussed as pivotal factors in the prognosis of heart failure. The authors posited that in patients with abdominal obesity, an elevation in intra-abdominal pressure during the respiratory cycle could result in a diminution of CRF. and the obesity paradox in cardiovascular disease, including HF, is noted primarily in individuals with low CRF[21–23]. Previous research has demonstrated that among 98 confirmed COVID-19 patients, those with a higher TG/HDL-C ratio were more susceptible to cardiac injury and heart failure[24]. Consequently, drawing upon our research findings and considering the obesity paradox associated with WHtR, we reasonably propose that WHtR exceeding

a certain threshold may be inversely associated with the incidence of HF. Given the association between obesity and a multitude of diseases, such as cardiovascular diseases, type 2 diabetes, obstructive sleep apnea, asthma, pulmonary embolism, osteoarthritis, cancer, and liver diseases[25–28], it is noted that patients tend to exhibit multiple comorbidities rather than solely HF when the CMI exceeds 6.49. This may elucidate the emergence of a plateau phase as well. Furthermore, when the value of the CMI exceeds approximately 20, there appears to be a renewed positive correlation with HF, although due to the scant sample size within this range, the conclusion lacks robustness.

All constituents of the CMI are integral components of metabolic syndrome[29,30], which encompasses central obesity, insulin resistance, dyslipidemia, and hypertension—factors prevalent in cardiovascular diseases and heart failure[31,32]. Obesity, crucial in metabolic syndrome, increases cardiac workload and promotes an inflammatory state harmful to heart function[33]. Insulin resistance exacerbates this by decreasing glucose uptake and increasing free fatty acid metabolism, leading to heart dysfunction. The relationship between metabolic syndrome and heart failure is also reflected in the modulation of biomarkers such as leptin, IL-1ra, and FABP-4, highlighting the roles of adiposity, inflammation, and lipid signaling[34]. Furthermore, obesity and ectopic fat deposition, including in the heart, heighten cardiometabolic risk and cause structural cardiac changes, such as increased left ventricular mass and impaired diastolic function, which predispose to heart failure[35]. Recent studies show adipose tissue, actively involved in metabolic regulation, maintains a two-way communication with the cardiovascular system, including both endocrine and paracrine interactions, allowing it to respond adaptively to cardiovascular signals[36].

TG/HDL-C ratio is a significant predictor of coronary artery disease[8,9], and the CMI, which amalgamates features of adiposity and lipidemia, serves to discern the risk of diabetes[6]. Type 2 Diabetes Mellitus (T2DM), a major HF risk[17,18], involves Advanced Glycation End-products (AGEs), oxidative stress, inflammation, and endothelial dysfunction, leading to atherosclerosis, hypertension, and dyslipidemia[35,37]. These conditions foster CAD through vulnerable plaque formation, increasing acute myocardial infarction and HFrEF risks[38]. T2DM also underlies diabetic cardiomyopathy (DCM), wherein microvascular coronary artery disease is a significant factor[39,40], characterized by cardiomyocyte insulin resistance, altered glucose and fatty acid metabolism, reducing cardiac function and causing lipotoxicity[41,42]. Addressing glycemic control and metabolic factors like lipid imbalances is crucial for HF prevention in diabetic patients[43]. Regrettably, the NHANES does not accommodate the stratification of heart failure subtypes within our analytical framework. Nonetheless, the results procured remain meaningful, suggesting that the aforementioned factors may exert influences of varying magnitudes across distinct subtypes of heart failure.

An elevation in BMI is associated with an enhanced risk for heart failure[44]. Nevertheless, CMI possesses a more pronounced ability to discern heart failure compared to BMI, and equally facile in computation. Consequently, incorporating CMI into standard clinical practice could refine the detection of individuals at heightened risk for heart failure, thereby facilitating timely interventions and potentially yielding superior

health outcomes. Looking ahead, the role of CMI in heart failure management is promising, necessitating further confirmation of its prognostic utility across various populations and healthcare environments. Delving into the specific mechanisms by which CMI influences heart failure could lead to personalized treatment approaches, reducing the heart failure risk among individuals with elevated CMI levels. Prospective studies are essential to validate these observations and elucidate the causal pathways.

Our study boasts several merits. Utilizing data from ten NHANES cycles, we captured a broad spectrum of the U.S. adult population, enhancing the generalizability of our findings. The ample sample size enabled detailed subgroup investigations. We employed weighted adjustments and controlled for confounders, mitigating sampling biases and ensuring the validity of our conclusions. Moreover, we investigated the non-linear interplay between the CMI and HF, identifying a potential threshold in this relationship, with the curve fitting aligning with regression analyses, confirming the reliability of our outcomes. However, certain limitations warrant mention. The cross-sectional format restricts our ability to infer causality between CMI and HF. The transferability of our results may be influenced by diverse genetic, lifestyle, and environmental contexts across populations. Furthermore, NHANES database limitations hindered deeper stratified analyses and comprehensive subgroup examinations, preventing a thorough exploration of the CMI-HF correlation across various HF types. Despite adjusting for multiple confounders, residual confounding effects cannot be entirely ruled out.

5. Conclusion

Our research indicates that the level of CMI bears a substantial positive correlation with the incidence risk of HF, with the relationship between CMI and HF being non-linear. Additionally, the CMI is a better predictor of HF than the BMI. Further investigation into the underlying causes is necessary, and more extensive forward-looking research is needed to confirm these observations.

Declarations

Ethical Approval

The National Center for Health Statistics Institutional Review Board approved all study procedures. Since 1999, this survey, which represents a cross-section of the population, has been published online every two years. Public datasets are available at https://www.cdc.gov/nchs/nhanes/index.htm. The study utilized publicly available deidentified data and did not require explicit informed consent. Ethical approval or individual permissions were not sought for this study, as it utilized a publicly accessible database.

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Tables

Table 1. Weighted comparison in basic characteristics.

	Overall(n=22042	HF n=706	Non-HF n=21336	P value
Age(years)	47.32 ± 16.67	65.30 ± 13.38	46.87±16.49	<0.001
Family poverty income ratio	2.96 ± 1.59	2.30 ± 1.40	2.97 ± 1.59	<0.001
BMI (kg/m2)	28.67 ± 6.61	30.94 ± 7.10	28.62 ± 6.59	<0.001
TG (mmol/L)	1.49 ± 1.34	1.81 ± 1.59	1.49 ± 1.33	<0.001
TC (mmol/L)	5.05 ± 1.08	4.74 ± 1.16	5.06 ± 1.07	<0.001
HDL- C(mmol/L)	1.39 ± 0.42	1.26 ± 0.39	1.39 ± 0.42	<0.001
LDL- C(mmol/L)	3.00 ± 0.90	2.70 ± 0.94	3.01 ± 0.90	<0.001
Waist circumference(cm)	98.38 ± 16.31	107.91 ± 16.81	98.15 ± 16.23	<0.001
Height(cm)	169.01 ± 10.05	167.81 ± 11.01	169.04 ± 10.02	0.0056
WHTR	0.58 ± 0.10	0.64 ± 0.10	0.58 ± 0.10	<0.001
CMI	0.79 ± 1.08	1.17 ± 1.83	0.78 ± 1.05	<0.001
Gender (%)				0.0356
Male	49.28	53.8	49.17	
Female	50.72	46.2	50.83	
Race/Ethnicity (%)				0.0012
Mexican American	8.18	4.1	8.28	
Other Hispanic	5.61	4.39	5.64	
Non-Hispanic white	69.06	73.89	68.95	
Non-Hispanic black	10.45	12.43	10.4	
Other race or multi-racial	6.69	5.2	6.73	
Education level (%)				<0.001
Less Than 9th Grade	6.12	13.79	5.93	
9-11th Grade	11.37	19.54	11.17	
High School Grad	24.04	27.74	23.94	
Some College	30.63	25.76	30.75	
College Graduate or above	27.85	13.17	28.21	
Hypertension (%)				<0.001

Yes	31.15	73.57	30.11	
No	68.85	26.43	69.89	
Diabetes (%)				<0.001
Yes	8.7	34.78	8.06	
No	91.3	65.22	91.94	
Coronary heart disease (%)				<0.001
Yes	3.46	37.44	2.62	
No	96.54	62.56	97.38	
Angina pectoris (%)				<0.001
Yes	2.37	25.71	1.79	
No	97.63	74.29	98.21	
Heart attack (%)				<0.001
Yes	3.38	43.48	2.4	
No	96.62	56.52	97.6	
Stroke (%)				<0.001
Yes	2.69	20.33	2.25	
No	97.31	79.67	97.75	
Smoked at least 100 cigarettes (%)				<0.001
Yes	47.12	61.17	46.77	
No	52.88	38.83	53.23	

Mean ± SD for continuous variables: P value was calculated by weighted linear regression model. % for categorical variables: P value was calculated by weighted chi-square test. BMI, body mass index; TG, triglyceride; TC, total cholesterol; HDL-C, high-density lipoprotein Cholesterol; LDL-C, low-density lipoprotein Cholesterol; WHtR, waist-to-height ratio; CMI, cardiometabolic index.

Table 2. Association of CMI with HF and stratified logistic regression.

	Model 1 OR (95%CI) P-value	Model 2 OR (95%CI) P-value	Model 3 OR (95%CI) P-value
CMI	1.15 (1.11, 1.19) <0.001	1.17 (1.13, 1.22) 0.001	1.13 (1.07, 1.17) <0.001
stratified by gender			
Male	1.11 (1.06, 1.15) <0.001	1.14 (1.08, 1.20) <0.001	1.10 (1.04, 1.16) <0.001
Female	1.25 (1.10, 1.43) <0.001	1.30 (1.11, 1.51) 0.0014	1.16 (1.08, 1.24) <0.001
stratified by race			
Mexican American	1.09 (0.999, 1.18) 0.051	1.10 (0.99, 1.22) 0.089	0.96 (0.74, 1.23) 0.731
Other Hispanic	1.07 (0.99, 1.15) 0.103	1.08 (0.98, 1.18) 0.112	1.06 (0.90, 1.26) 0.494
Non-Hispanic white	1.17 (1.11, 1.24) <0.001	1.19 (1.12, 1.27) <0.001	1.13 (1.08, 1.19) 0.001
Non-Hispanic black	1.41 (1.21, 1.65) <0.001	1.54 (1.29, 1.84) <0.001	1.24 (1.03, 1.50) 0.029
Other race or multiracial	1.30 (1.11, 1.51) 0.0012	1.29 (1.05, 1.58) 0.018	1.31 (1.09, 1.59) 0.005
stratified by education			
Less Than 9th Grade	1.03 (0.95, 1.11) 0.454	1.06 (0.97, 1.15) 0.218	1.00 (0.89, 1.13) 0.957
9-11th Grade	1.13 (1.06, 1.21) <0.001	1.17 (1.08, 1.27) <0.001	1.15 (1.08, 1.23) <0.001
High School Grad	1.15 (1.06, 1.26) 0.0018	1.24 (1.12, 1.37) <0.001	1.13 (0.98, 1.30) 0.096
Some College	1.16 (1.08, 1.25) <0.001	1.18 (1.08, 1.28) <0.001	1.11 (1.01, 1.22) 0.031
College Graduate or above	1.18 (1.07, 1.31) 0.0016	1.18 (1.07, 1.31) <0.001	1.18 (1.07, 1.29) <0.001
stratified by hypertension			
Yes	1.15 (1.08, 1.22) <0.001	1.21 (1.13, 1.30) <0.001	1.150 (1.08, 1.22) <0.001
No	1.09 (1.05, 1.14) <0.001	1.10 (1.05, 1.16) <0.001	1.08 (0.999, 1.17) 0.055
stratified by diabetes			

Yes	1.08 (1.02, 1.14) 0.0057	1.14 (1.07, 1.21) <0.001	1.14 (1.08, 1.20) <0.001
No	1.12 (1.07, 1.17) <0.001	1.14 (1.08, 1.19) <0.001	1.11 (1.04, 1.18) 0.0014
stratified by coronary heart disease			
Yes	1.26 (1.02, 1.56) 0.034	1.29 (1.03, 1.62) 0.027	1.22 (1.01, 1.49) 0.046
No	1.14 (1.10, 1.17) <0.001	1.16 (1.12, 1.21) <0.001	1.11 (1.07, 1.16) <0.001
stratified by angina			
Yes	1.10 (0.88, 1.37) 0.410	1.15 (0.91, 1.47) 0.248	1.20 (0.995, 1.46) 0.061
No	1.14 (1.10, 1.18) <0.001	1.17 (1.12, 1.21) <0.001	1.12 (1.08, 1.16) <0.001
stratified by heart attack			
Yes	1.15 (1.16, 1.58) <0.001	1.41 (1.20, 1.65) <0.001	1.39 (1.16, 1.66) <0.001
No	1.13 (1.09, 1.17) <0.001	1.16 (1.11, 1.20) <0.001	1.09 (1.03, 1.14) 0.0014
stratified by stroke			
Yes	0.998 (0.85, 1.17) 0.985	1.03 (0.87, 1.23) 0.725	0.95 (0.76, 1.18) 0.625
No	1.16 (1.11, 1.20) <0.001	1.18 (1.13, 1.23) <0.001	1.13 (1.09, 1.18) <0.001
stratified by smoke status			
Yes	1.11 (1.06, 1.16) <0.001	1.14 (1.08, 1.20) <0.001	1.10 (1.04, 1.15) <0.001
No	1.18 (1.11, 1.25) <0.001	1.21 (1.13, 1.31) <0.001	1.17 (1.09, 1.25) <0.001

Model 1: no covariates were adjusted. Model 2: age, gender, and race were adjusted. Model 3: age, gender, race, educational level, family poverty income ratio, hypertension, diabetes, coronary heart disease, angina, heart attack, stroke, and smoking status.

Table 3. Analysis of threshold effect and saturation effect.

CMI	Adjusted OR (95% CI), P value
Model I	
A straight-line effect	1.12 (1.07, 1.17) <0.001
Model II	
Fold points (K)	6.49
< K-segment effect 1	1.26 (1.15, 1.37) 0.0016
> K-segment effect 2	1.02 (0.93, 1.12) 0.665
Effect size difference of 2 versus 1	0.81 (0.70, 0.94) 0.007
Equation predicted values at break points	-1.58 (-1.96, -1.21)
Log likelihood ratio tests	0.005

CI, confidence interval; CMI, cardiometabolic index; Weighted by: Full sample mobile examination center exam weight. Adjusted for age, gender, race, educational level, family poverty income ratio, hypertension, diabetes, coronary heart disease, angina, heart attack, stroke, and smoking status. OR represents the slope of the curve.

Table 4. Areas under the receiver operating characteristic curves of CMI and BMI for predicting HF occurrence.

Variables	Best threshold	Sensitivity	Specificity	AUC (95% CI)
CMI*	0.57	0.66	0.53	0.63(0.61,0.65)
BMI	27.15	0.68	0.46	0.59(0.57,0.61)

CMI, cardiometabolic index; BMI, body mass index.

*P < 0.001, DeLong test was used to compare the AUC of CMI and BMI.

Figures

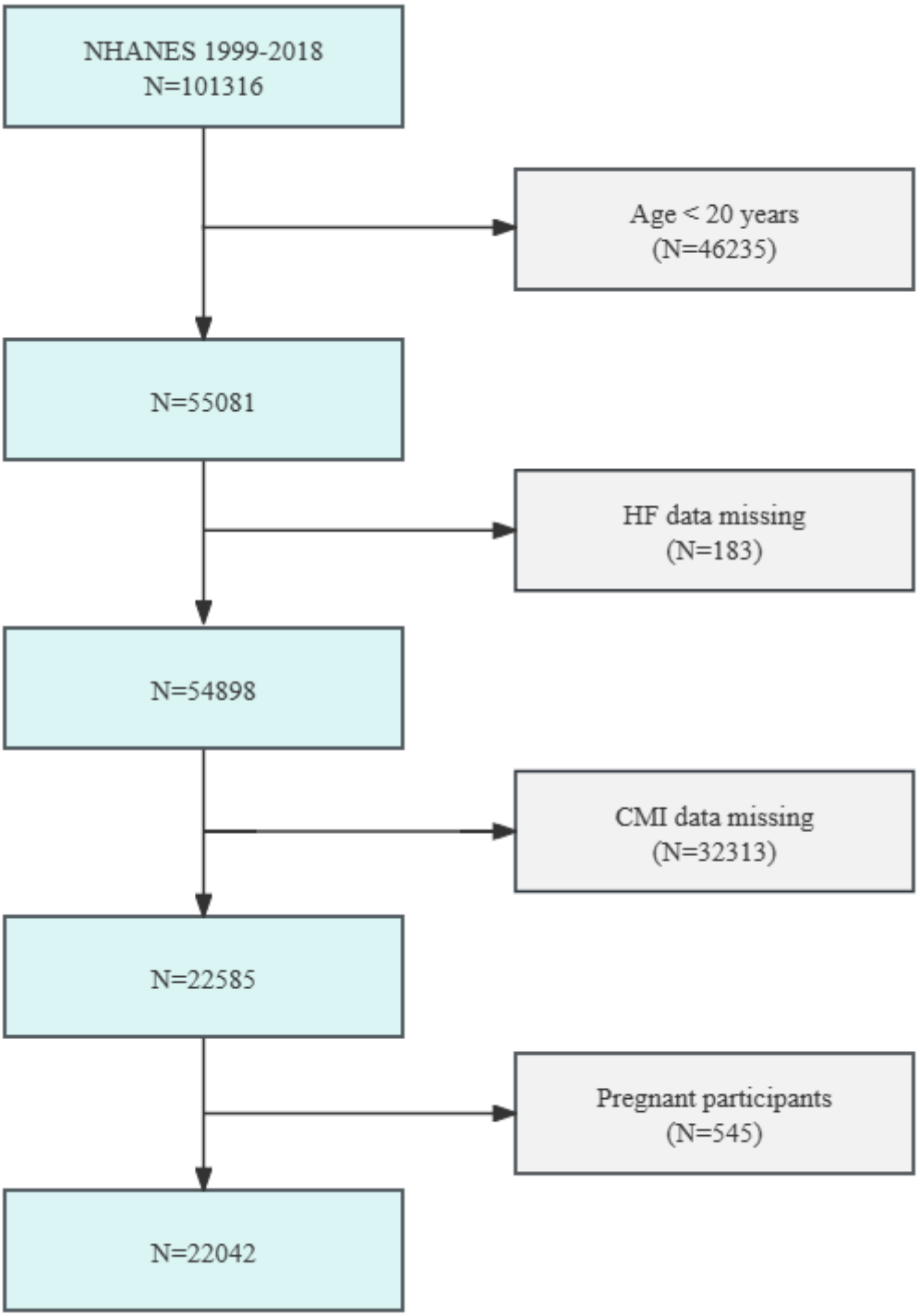

Figure 1

Flowchart of participant selection. NHANES, National Health and Nutrition Examination Survey; HF, heart failure; CMI, cardiometabolic index.

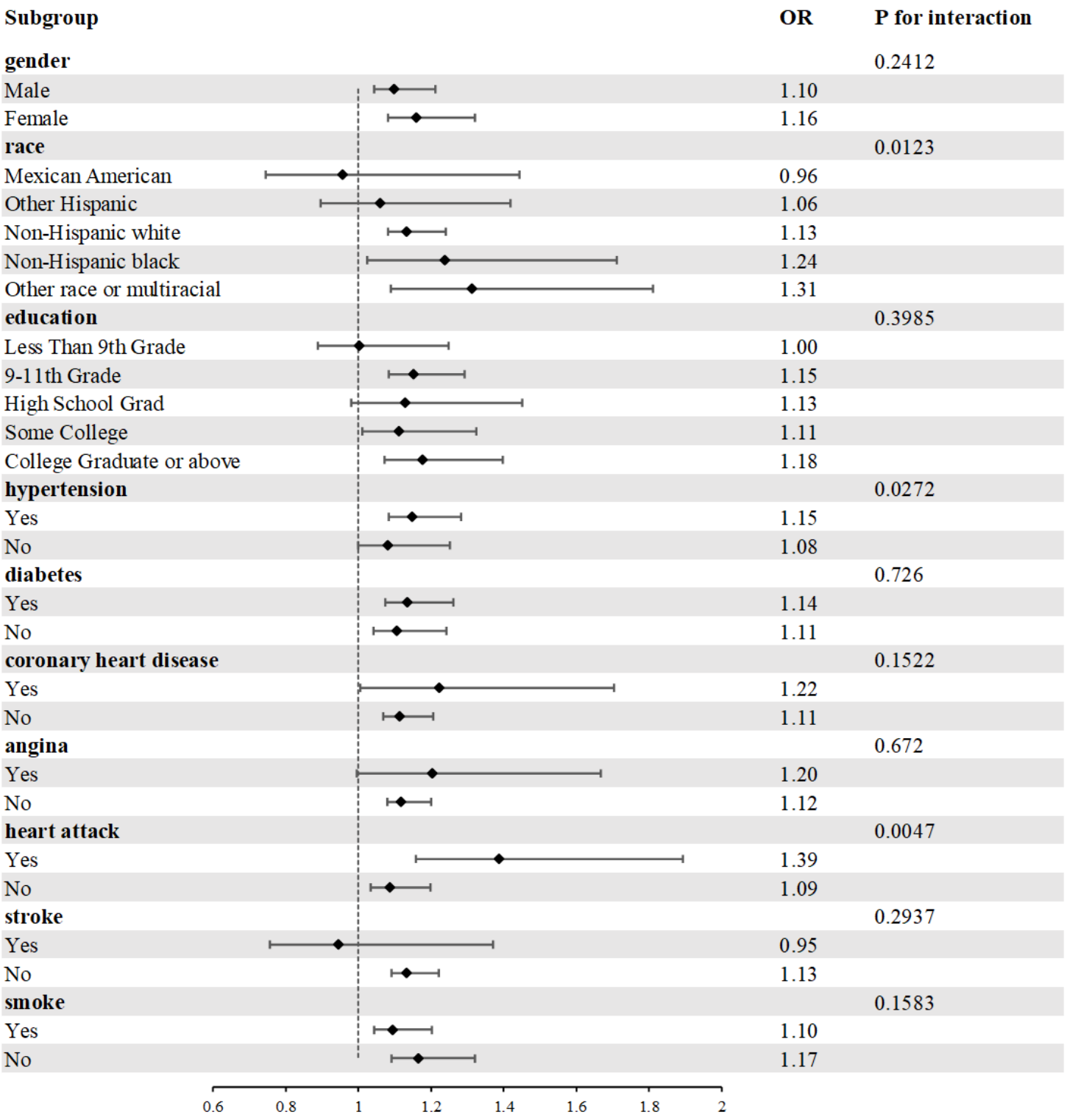

Figure 2

Subgroup analysis for the association between cardiometabolic index and heart failure.

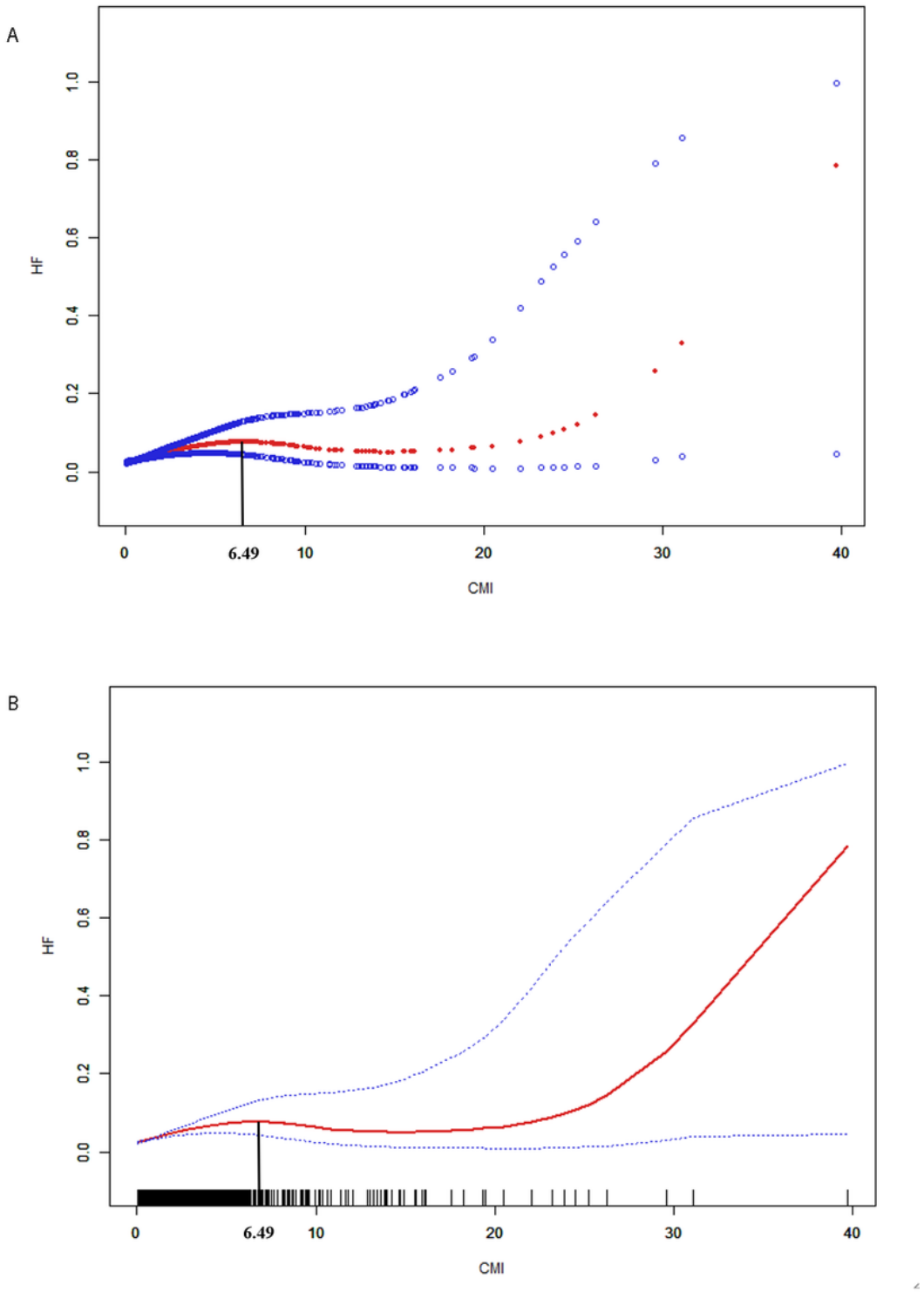

Figure 3

The association between cardiometabolic index and heart failure. The solid red line represents the smooth curve fit between variables. Blue bands represent the 95% confidence interval from the fit.

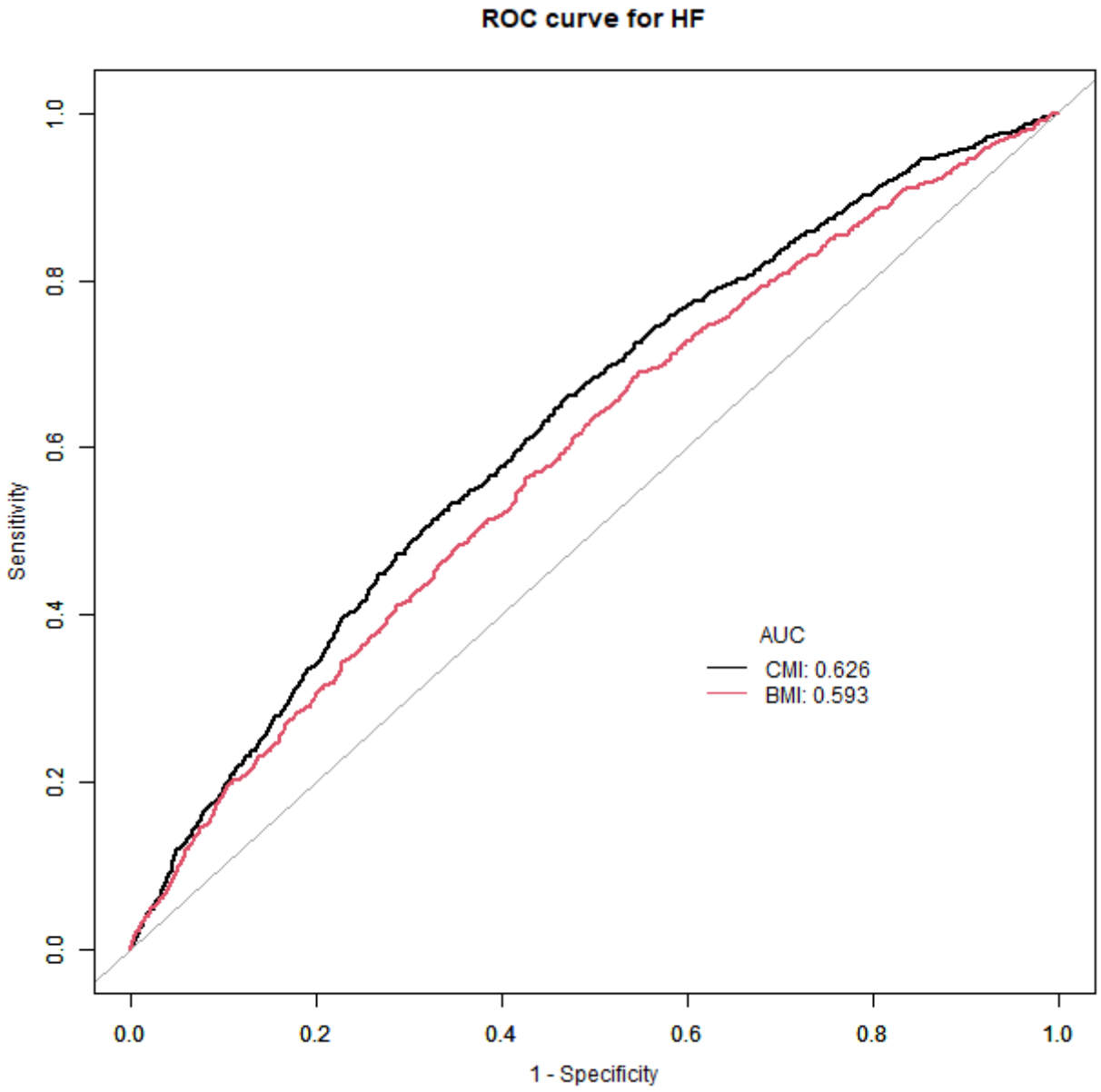

Figure 4

ROC curves of CMI and BMI for predicting HF occurrence